Surgical treatment of renal artery dissection in 25 patients: Indications and results

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Objective: **Results of surgical revascularization in 25 patients with renal artery dissection (RAD) over 14 years, with mean follow-up of 55.3 months (range, 10-111 months), were analyzed. Indications for surgery were renovascular hypertension and preservation or improvement of kidney function.**

Patients and Methods: **Two patients (both 20 years of age) underwent emergency surgery after severe trauma; 23 patients (mean age, 41 years) underwent elective surgery in a chronic stage of disease. Preoperative, postoperative, and follow-up examinations included duplex ultrasound scanning, determination of serum creatinine and urea concentrations, and evaluation of blood pressure control. All long-term patients underwent digital subtraction angiography preoperatively and postoperatively. All histologic specimens of resected renal arteries were re-evaluated by two independent pathologists.**

Results: **Histologic re-evaluation confirmed the traumatic origin in 2 patients who underwent emergency surgery and 1 who underwent elective surgery. Renal artery dissection developed spontaneously, with no histologic signs of trauma or fibromuscular dysplasia, in 22 patients. In 17 revascularized kidneys (61%) a kidney infarction had already developed preoperatively, and the kidneys were diminished in size or function. Results of revascularization and improvement of hypertension depended on preoperative extent of renal infarction. Hypertension resolved or improved in 86% of patients without preoperative kidney damage, but in only 38% with preoperatively damaged kidneys. Kidney function was preserved in 23 of 28 revascularized kidneys (82%). During follow-up, late renal artery occlusion developed in 3 kidneys.**

Conclusions: **Renal artery dissection can be effectively treated with surgical revascularization. Primary nephrectomy should be considered only in patients with a large ischemic kidney infarction, with significant deterioration of kidney function, to effectively cure or improve severe renovascular hypertension. (J Vasc Surg 2003;37:761-8.)**

Spontaneous renal artery dissection (RAD), first reported in 1944 by Bumpus et al,[1] is rare. In most patients this unusual disorder is discovered during diagnostic work-up because of recent increase in blood pressure in young persons. Nevertheless, some patients have flank pain at the acute stage of disease, probably due to thromboembolism and partial kidney infarction, and often urolithiasis is wrongly suspected.

Most reports have been isolated cases or small series of patients with RAD only, and to date surgical revascularization of RAD has been documented in only four larger series with a maximum of 22 patients each.[2-5] Because RAD is uncommon, its spontaneous course and the best therapeutic strategy are still under debate. We retrospectively evaluated indications and results in 25 patients, which represents the senior author's (W.S.) experience over 14 years.

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Competition of interest: none.

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PATIENTS AND METHODS

Study population

From March 1987 to August 2000, of 680 patients who underwent renal artery reconstruction (atherosclerotic lesions, n = 550; fibromuscular dysplasia, n = 105) 25 patients with primary RAD were treated surgically at our institution. During the same period 4 additional patients with RAD caused by radiologic intervention to treat an atherosclerotic lesion and 10 patients with RAD due to aortic dissection underwent reconstructive surgery. Because of the different causes, we did not consider these two subgroups for our sample. The group with primary RAD consisted of 7 women and 18 men. Two 20-year-old patients (1 woman, 1 man) underwent emergency surgery after severe trauma; the other 23 patients, with mean age 41 years (range, 31-56 years) underwent elective surgery in a chronic stage of disease.

Three of 25 patients had bilateral RAD, and each of the two kidneys had 2 dissected main renal arteries. Thus 25 patients had 28 affected kidneys with 30 dissected renal arteries, in which reconstruction was indicated. Two additional kidneys contralateral to those with dissected renal arteries were revascularized simultaneously because of critical stenosis, which was detected at angiography and suspected to be caused by fibromuscular dysplasia.

Histologic re-evaluation

For this retrospective study, all renal artery specimens were re-examined by two experienced independent pathol-

Table I. Symptoms of chronic renal artery dissection (n = 23)

Symptom	*Patients*	
	n	*%*
Hypertension	21	91
Flank pain	9	39
Abdominal pain	2	9
Creatinine >1.5 mg/dL	3	13

ogists to identify the cause of the dissection. For diagnosis of fibromuscular dysplasia, the criteria of Harrison and colleagues were used.[6,7] Trauma as the underlying cause of dissection was considered if one or more of the following histopathologic features, mainly in the adventitia, were observed: extensive bleeding or distinct tears in the adventitia; siderin deposits, indicating older bleeding into the adventitia; and fresh bleeding, partly in the direct area of small capillaries (Fig 1). Specimens showing none of these traumatic features or any hint of fibromuscular dysplasia as the underlying cause were classified as idiopathic dissections (Fig 2).

Diagnostic work-up

Preoperative examination. In all patients preoperative B-mode ultrasound was performed to evaluate the size of the kidneys. RAD was diagnosed at angiography in all elective cases. For evaluation of kidney function, blood creatinine and urea concentrations were measured. More recently, duplex scanning and measurement of intraparenchymal resistance indexes have been included in the preoperative diagnostic work-up. In some patients additional examinations included isotope renography (10 patients) and differential bilateral renal vein renin assays (2 patients).

Postoperative examination. Surgical results were determined with measurement of blood pressure and blood creatinine and urea concentrations. Postoperative angiography during hospital stay was performed in all patients but one, who refused the procedure, but in this patient the dissected renal artery had already become occluded during surgery, and later duplex scanning did not reveal any renal perfusion.

Follow-up. Follow-up examination included blood pressure measurements, laboratory studies, and duplex ultrasound scanning in 21 patients with 24 reconstructed kidneys. One patient, in whom the reconstructed kidney became occluded soon after surgery, was lost to follow-up. In another 3 patients only clinical information, ie, blood pressure, medications, and laboratory findings, could be obtained. These 3 patients had undergone surgery successfully, and early postoperative angiography demonstrated an open renal artery. Eight patients underwent late angiography.

Fig 1. Transverse section of a renal artery with traumatic dissection in a 19-year-old man after attempted suicide by jumping out of a window. Note extensive bleeding within the adventitia (*asterisk*), complete rupture of media and both elastic laminae, and rupture of the opposite lamina elastica externa (*arrow*) (elastic-van Gieson stain; original magnification ×40).

Definition of blood pressure change and status of preoperative kidney damage

Blood pressure at admission served as the reference point for determining the results of surgery. Subsequently, blood pressure was assessed at discharge from the hospital and during follow-up 55.3 ± 32.8 months (range, 10-111 months) after surgery. Hypertension was estimated if blood pressure was 140/90 mm Hg or higher[8] or if antihypertensive drugs were required. Postoperatively, cured hypertension was defined as blood pressure less than 140/90 mm Hg without antihypertensive drugs. Conditions were considered improved if medications required preoperatively for blood pressure control could be reduced by at least one drug or if systolic blood pressure decreased by at least 20 mm Hg and diastolic blood pressure decreased by at least 10 mm Hg with unchanged drug administration before and after surgery. Conditions not meeting these criteria for cure or improvement were categorized as unchanged or deteriorated if the patient required additional antihypertensive drugs to stabilize blood pressure.

Kidneys were considered preoperatively damaged if they met one of the following criteria: length less than 10 cm; renal infarction seen on angiograms, CT scans, or duplex ultrasound scans; function of the affected kidney less than 40% of that of the contralateral unaffected kidney at isotope renography.

Statistical analysis

All medical and surgical records and imaging studies were reviewed carefully. For calculation of significance, the Fisher exact test was used.

Table II. Influence of surgery on hypertension

No. of antihypertensive drugs before surgery	*No. of patients*	*Hypertension at discharge*			*Hypertension at follow-up*			*Months of follow-up (±SD)*
		Status	*n*	*%*	*Status*	*n*	*%*	
0 (Untreated hypertension)	3	Cured	1		Cured	1		
		Improved	0		Improved	0		
		Unchanged	2		Unchanged	2		55.3 ± 17.9
		Worse	0		Worse	0		
1-2	14	Cured	4		Cured	2		
		Improved	4		Improved	3		
		Unchanged	5		Unchanged	8		65.4 ± 31.5
		Worse	1		Worse	1		
3 or more	4	Cured	0		Cured	0		
		Improved	2		Improved	2		
		Unchanged	2		Unchanged	1		20.8 ± 11.5
		Worse	0		Worse	0		
					Patient dead	1		
All patients	21	Cured	5	24	Cured	3	14	48.3 ± 5.5
		Improved	6	29	Improved	5	24	59.4 ± 40.6
		Unchanged	9	43	Unchanged	11	52	54.6 ± 34.4
		Worse	1	5	Worse	1	5	86
					Patient dead	1	5	36

Twenty-one of 25 patients with acute and chronic renal artery dissection had hypertension preoperatively.

Clinical findings

Symptoms. Hypertension was the main symptom of RAD. Twenty-one of 23 patients (91%) with chronic RAD had elevated blood pressure (Table I). The two patients with acute dissection did not have hypertension before emergency revascularization. On average, 1.7 different antihypertensive drugs were necessary to achieve blood pressure control (Table II). Whereas 12 dissections developed silently, 9 of 23 patients (39%) with chronic RAD had colic within the flank at the onset of disease, and 2 additional female patients had abdominal discomfort caused by simultaneous dissection of renal and visceral arteries (1 coeliac trunk, 1 superior mesenteric artery; Table I). Abdominal discomfort probably developed at the onset of acute RAD at a mean of 4.7 months (range, 1-14 months; median, 3 months) before surgery. Three patients (13%) had renal insufficiency with serum creatinine concentration greater than 1.5 mg/dL (Table I).

Additional disease. Two patients had non-insulin-dependent diabetes mellitus. Two female patients had contralateral renal artery stenosis, which was repaired simultaneously. Because of findings at preoperative angiography, the stenosis was suspected to be caused by fibromuscular dysplasia. In a 47-year-old woman, abdominal aortic coarctation was the underlying cause of RAD. Of interest, 4 of 25 patients (16%) had concomitant dissection in other vascular regions: visceral arteries (celiac trunk and superior mesenteric artery, respectively) in 2 female patients; internal carotid artery, which did not require reconstruction, in 1 male patient; and common iliac artery, surgically treated, in 1 patient. Although drug abuse, especially with cocaine or methamphetamine, is associated with dissection, we did not find this in any of our patients.

Preoperative kidney status. According to kidney function and structure, 17 of 28 reconstructed kidneys (61%) in 17 patients (68%) showed signs of preoperative damage (Fig 3). Fourteen patients had experienced kidney infarction, probably due to branch renal artery dissection and thromboembolism in 13 patients and to thromboembolism from a renal mainstem aneurysm in 1 patient. In 1 patient, contrast-enhanced renography revealed a considerably reduced kidney length (8 cm), with severely reduced renal function of unknown origin. One patient underwent emergency surgery 10 hours after a traumatic accident. CT scans obtained immediately before the operation did not show any kidney perfusion on the affected side. Another patient underwent emergency surgery 2 days after a traumatic accident. Preoperative angiograms revealed a nearly total mainstem occlusion, and intravenous pyelograms did not show any excretion.

Table III. Operative techniques in renal artery reconstruction

Technique	*Renal arteries*	
	n	*%*
Resection, vein interposition graft	25	83
Resection, end-to-end anastomosis	2	7
Aneurysm resection	1	3.3
Intraoperative dilation	1	3.3
Vein patch plasty	1	3.3
Total	30	100

Twenty-five patients had 28 affected kidneys, with 30 dissected renal arteries.

Radiologic findings. Typical radiologic findings were long irregular stenoses of the renal artery, which, in contrast to atherosclerotic lesions, developed at a certain distance to the aorta (Fig 3). In 9 kidneys dissection extended into renal branch arteries, and in 5 additional kidneys some

Table IV. Influence of preoperative kidney damage on hypertension at discharge

Kidneys preoperatively	*No of patients*	*Hypertension cured or improved*		*Hypertension unchanged or deteriorated*		*P*
		n	*%*	*n*	*%*	
Undamaged	7	6	86	1	15	.058
Damaged	13	5	38	8	62	
Dissected kidney undamaged, but contralateral kidney* disturbed	1			1		
Total	21					

Twenty-one of 23 patients with chronic RAD had hypertension preoperatively. Two patients with acute traumatic dissection did not have hypertension before operative intervention.

Kidneys were considered preoperatively damaged if they met one of the following criteria: length <10 cm; renal infarction seen on angiogram, CT scan, or duplex scan; function of kidney with RAD compared with contralateral unaffected kidney <40% at isotope renography.

*Small and disturbed.

of the renal branch arteries were already occluded, probably from previous thromboembolism. Post-stenotic dilatation was common, and in 6 patients an isolated aneurysm developed near the renal hilus. In 4 patients fibromuscular dysplasia as the underlying cause was suspected at angiography. The renal arteries exhibited areas of luminal narrowing alternating with dilated segments greater in diameter than the normal renal artery.[9]

Surgical treatment

Indications for operative intervention. Surgery to treat renal artery dissection had two main goals: organ preservation and cure or improvement of renovascular hypertension. Simultaneous vascular reconstruction in various regions was indicated in 5 female patients and included reconstruction of the contralateral stenotic renal artery (n = 2), abdominal aortic coarctation (n = 1), and visceral artery dissection (n = 2).

Choice of surgical procedure. Revascularization was performed in all patients; none underwent primary total or partial nephrectomy. For prolongation of ischemic tolerance, flush perfusion with 1 L of 4°cold Ringer solution containing 20 mg of prostaglandin E and 1000 IU of heparin was administered continuously through a catheter in the renal main stem at the onset of renal ischemia.

Twenty-five dissected renal arteries were resected and replaced with an aortorenal bypass graft composed of greater saphenous vein taken from the groin (Fig 4). Other procedures consisted of local resection and end-to-end anastomosis (n = 2), aneurysm excision with tailoring (n = 1), intraoperative dilation (n = 1), and vein patch plasty (n = 1) (Table III). Because of distal extension of dissection, renal branch arteries were reconstructed in 13 (52%) patients as well, either with replacement of segmental arteries with a vein graft (n = 7) or reimplantation of renal branch arteries into a vein graft (n = 6). In 1 kidney ex situ reconstruction was necessary to facilitate the operative procedure. This patient required repair of the renal main stem and two other branch renal arteries.

Treatment of the contralateral nondissected renal artery stenosis consisted of intraoperative transluminal dilatation with dilators (n = 1) or resection and vein interposition grafting (n = 1).

RESULTS

Histologic findings

Renal artery specimens for histologic reevaluation were available in 24 cases. The traumatic origin was confirmed in 3 cases, including 2 acute and 1 chronic dissection (Fig 1). No sign of fibromuscular dysplasia was found in these specimens. Dissection in 21 cases was idiopathic and developed spontaneously, without any recognizable reason (Fig 2). In some specimens of typical dissection, neointimal proliferation approximately 1 mm of thickness was observed about 1 to 2 months after the dissecting event. This finding was similar to restenosis often seen after percutaneous transluminal coronary angioplasty.

Early results after surgery

Early postoperative complications. Postoperative complications developed in 5 of 25 patients (20%). Three renal arteries became occluded soon after surgery, but patency was restored with thrombectomy in 2 arteries. A herald abdominal bleeding in 1 patient was managed successfully. Inferior vena cava thrombosis developed in 1 patient, and thrombectomy of the inferior vena cava was unsuccessful.

Blood pressure control. Resolution of or improvement in hypertension depended on success of vascular reconstruction and extent of renal infarction. At discharge, hypertension had resolved in 5 patients (24%), improved in 6 patients (29%), was unchanged in 9 patients (43%), and was worse in 1 patient (5%) (Table II). Hypertension resolved or improved in 86% of patients with preoperative undamaged kidneys, but remained unchanged or worsened in 62% of patients with preoperative damaged kidneys (Table IV).

Blood pressure control was achieved in 11 of 17 patients (65%), whose reconstructed kidneys maintained perfusion postoperatively, whereas hypertension remained un-

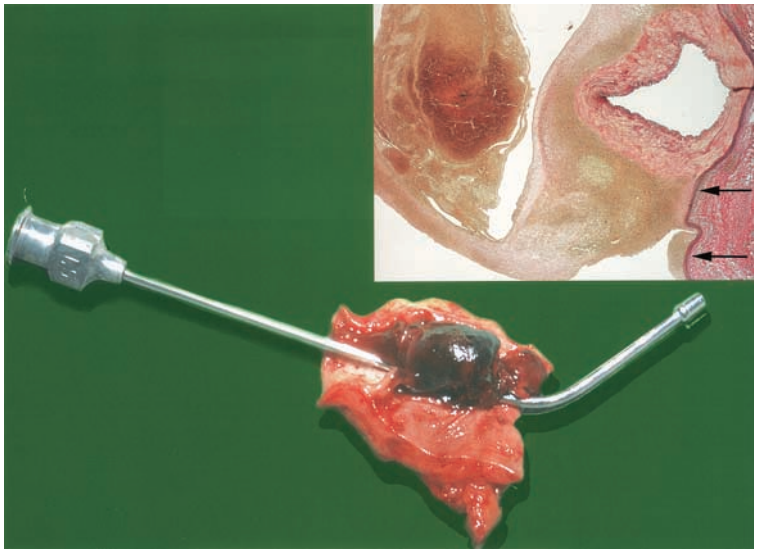

Fig 2. Resected renal artery. Canula is placed in the stenosed "true lumen" of the artery, and the aneurysmal "false lumen" is filled with fresh thrombus. *Inset,* Transverse section of the same specimen shows typical dissection with tear of the outer third of the media and extensive, partly organized bleeding. Note uninjured lamina elastica interna *(arrows)* (elastic-van Gieson stain; original magnification ×40).

changed or worsened in all 4 patients with loss of renal perfusion.

Postoperative kidney perfusion and organ preservation. Postoperative angiography and duplex ultrasound scanning demonstrated occlusion in 6 of 30 reconstructed dissected renal arteries (20%). Five of 28 revascularized kidneys (18%) in 4 patients had severely reduced or no renal perfusion (Fig 5). Although the renal artery in 5 kidneys after surgery was not patent, preoperative and postoperative creatinine and urea concentrations were the same in all but 1 patient. Renal failure developed in 1 patient with bilateral RAD in whom the reconstructed arteries became occluded soon after surgery, and permanent dialysis was required after surgery.

Late results after surgery.

Mean follow-up in 24 patients (96%) was 55.3 ± 32.8 months (range, 10-111 months). One patient with acute RAD had undergone reconstruction of the renal artery after severe trauma of suicidal intent. However, the renal artery was already occluded at discharge. This patient was lost to follow-up.

Late redo operation. Seven of 25 patients (28%) underwent repeat surgery. In 5 patients stenosis developed within the renal reconstruction, leading to worsened blood pressure control (fibrotic stenosis, n = 4; persistent dissection, n = 1). The primary renal artery reconstruction was replaced with a new saphenous vein interposition graft in 3 patients and with renal prosthetic interposition graft in 2 patients. One of these patients died with a functioning graft. Despite repeat exploration, the cause of death remained uncertain and was eventually attributed to massive septicemia. In 1 patient the kidney had shrunk substantially because of renal artery occlusion and was removed to treat hypertension. A kidney from a living related donor was transplated in the patient, requiring permanent dialysis

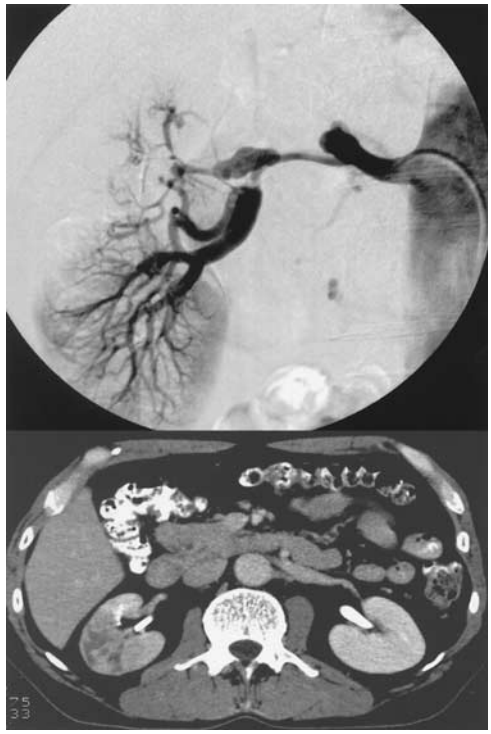

Fig 3. Preoperative digital subtraction angiogram and CT scan in a 46-year-old patient demonstrates dissection of the renal artery main stem. The right kidney has a renal infarction.

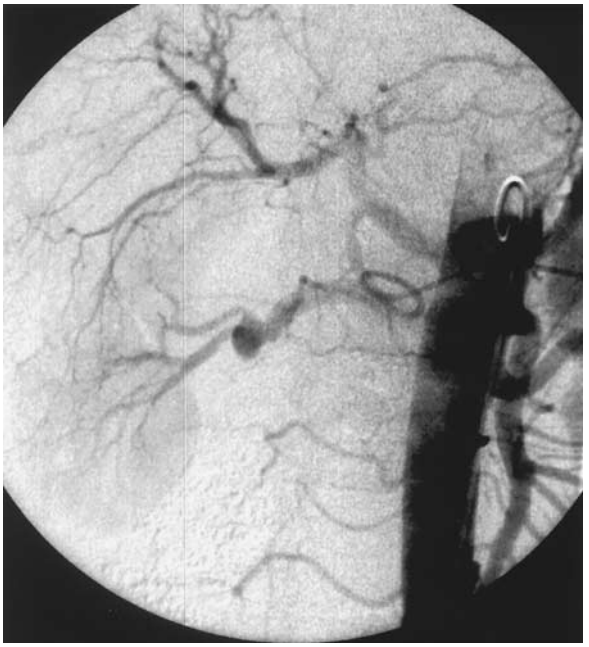

Fig 4. Postoperative digital subtraction angiogram of the same patient as in Fig 2. Dissected renal artery was resected and replaced with an aortorenal interposition graft with greater saphenous vein.

after surgery, and there were no further complications as normal renal function was achieved.

Blood pressure control. Hypertension resolved permanently in 3 patients (14%) and improved in 5 (24%), whereas in 12 patients (52%) hypertension remained more or less unchanged during follow-up. Progressive hypertension developed in 1 patient, and additional antihypertensive drugs were required for blood pressure control (Table II).

Late organ preservation and kidney perfusion. Duplex scans or angiograms obtained during follow-up demonstrated no or minimal blood perfusion in 8 of 25 reconstructed kidneys (32%), suggesting loss of function (Fig 5). Because of the small size of our sample, clear predictors of late renal artery occlusion could not be identified. Creatinine and urea concentrations remained unchanged from preoperative levels during follow-up in all patients. The function of the transplanted kidney remained stable during follow-up.

DISCUSSION

The pathogenesis of spontaneous dissection is unclear. Arterial dissection is initiated either by intramural hemorrhage, due to bleeding from the vasa vasorum, or by penetration of blood into the arterial wall through a primary intimal tear.[10,11]

Renal artery dissection has several similarities to carotid dissection, and some patients have both disorders simultaneously or subsequently,[12,13] as did one of our patients. Both entities affect middle-aged, otherwise healthy persons, and both dissection types have been observed with higher frequency in association with fibromuscular dysplasia, especially in women.[10-16] In our patients no histologic signs of fibromuscular dysplasia were detected in the resected renal arteries, although at angiographic evaluation fibromuscular dysplasia was suspected as the underlying cause of dissection in 4 patients. Two acute renal artery dissections were of traumatic origin, and prior trauma could be excluded in all but 1 chronic RAD. Most arterial dissections are idiopathic and develop spontaneously, probably in predisposed arteries.

Most RAD develop silently, without impressive symptoms at onset, and diagnostic work-up of hypertension reveals RAD already in a chronic stage. Other patients have symptoms similar to ureteral stone colic, and sometimes they are initially given treatment for urolithiasis. Measurement of serum lactic dehydrogenase, which provides a clue to renal infarction associated with RAD, may be helpful for differential diagnosis in the acute stage.

Because the diagnosis often is delayed, RAD is usually in the beginning of the chronic stage and the spontaneous course of acute dissection is unknown. Follow-up of patients with acute carotid dissection revealed a benign course with a high rate of spontaneous healing.[17,18] During follow-up only 15% of patients had persistent high-grade carotid stenosis or aneurysm that required surgical treatment.[17] Mori et al[19] reported the same course in a 33-year-old man with dissection of both renal arteries treated medically. Follow-up arteriograms 1 year after the initial attack showed almost complete resolution of dissection, with only minimal residual stenosis of both main renal arteries and recanalization of the intrarenal arterial branches. Blood pressure had returned to normal. In acute RAD, medical treatment with anticoagulation therapy may be considered if angiograms do not indicate risk for renal artery occlusion. If angiograms reveal high-grade stenosis or an aneurysm filled with thrombi, renal artery repair should be performed to preserve kidney function.

In the chronic stage of RAD, patients are referred to a vascular surgical unit for repair of the dissected arteries, which are suspected to be the cause of hypertension and renal insufficiency. By this time more than half of patients have already experienced a renal infarction[2,3] due to thromboembolism from the dissected artery or to occlusion of a dissected renal branch artery from intramural hematoma. Careful preoperative investigation is necessary to confirm the renovascular origin of hypertension caused by the dissected kidney. Although validation studies have reported high sensitivity and specificity for helical CT[20] and magnetic resonance angiography[21] in detecting significant renal artery stenosis, we believe selective intra-arterial digital subtraction angiography is necessary to evaluate the various morphologic aspects of RAD and to plan treatment strategy. In contrast to atherosclerotic lesions, the dissecting process starts distally from the aortic ostium. Furthermore, in about half of all cases the dissecting process also affects the intrarenal arterial branches, and sometimes accessory pol arteries are dissected also. For that reason even duplex ultrasound scanning cannot replace digital subtraction an-

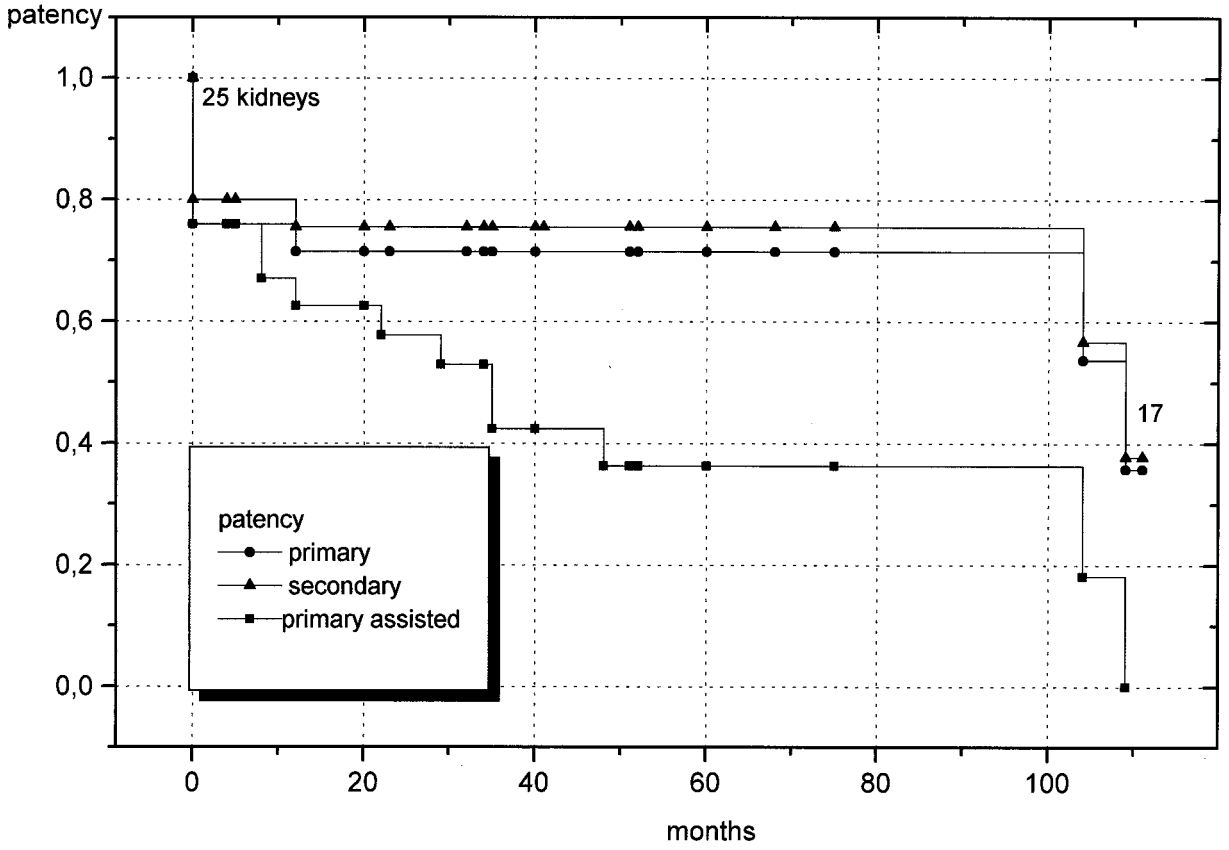

Fig 5. Kaplan-Meier analysis of patency rates after kidney revascularization to treat renal artery dissection. Duplex scans or angiograms were obtained during follow-up in 21 patients with 24 dissected and reconstructed renal arteries. In 1 additional patient, who was lost to follow-up, the reconstructed artery became occluded directly after surgery. *Patency: primary,* uninterrupted, with no procedure performed; *secondary,* patency with graft thrombectomy; *primary assisted,* patency never lost but maintained with prophylactic intervention to treat graft stenosis.

giography. Performed by experienced investigators, this technique is highly accurate for measuring the main renal artery, but fails in evaluation of aberrant renal arteries.[22] We believe duplex scanning is reliable for evaluation of kidney parenchymal perfusion, and combined with measurement of the main stem velocity and intraparenchymal resistance, evaluation of the hemodynamics of renal artery stenosis seems possible. For that reason, our routine preoperative renal investigation consists of digital subtraction angiography, duplex scanning, and isotope renography to evaluate bilateral renal function. Renal vein renin measurements may be helpful in identifying the kidney responsible for renin-dependent hypertension.[23]

Revascularization of dissected renal arteries requires some surgical expertise because of extension into branch vessels and perivascular inflammation and scarring of the diseased arterial segment. Extracorporeal repair with autotransplantation has been recommended in complex branch involvement,[2,3,24,25] but could be averted in all but one of our patients because flush perfusion with 4° C cold Ringer solution containing heparin and prostaglandin was used to increase ischemic tolerance of the kidney. Resection of the diseased segment and replacement with autologous material (saphenous vein or autologous artery graft) is the preferred method of reconstruction.[2-5] In contrast to other authors, who performed primary nephrectomy in 16% to 40% of dissected kidneys,[2-5] we used revascularization in all of our patients to preserve kidney function. Furthermore, 20% of our patients had bilateral processes that had to be treated simultaneously. However, in 20% of our revascularization procedures reconstruction was not successful. At discharge, hypertension had resolved or was improved in more than half of our patients, but during follow-up only 38% had benefited permanently from renal revascularization. These results are worse than previous results of studies in which we reported surgical treatment of hypertension caused by fibromuscular dysplasia. Permanent resolution or improvement in hypertension was achieved in 67% of these patients.[26] A possible explanation for the lower success rate in patients with RAD might be the preoperative existence of renal parenchymal infarction, causing renoparenchymatous hypertension, which cannot be treated with revascu-

larization alone. Lacombe,[2] in his series of 22 patients with RAD, reported resolution or improvement of hypertension in about 90%, but in contrast to our sample, he performed primary nephrectomy in 6 patients (26%) and partial nephrectomy in 2 patients (9%). Lacombe reports that in his revascularization group all "cured" patients had normal anatomy, whereas patients in whom hypertension was only improved or unchanged had persistent changes in the kidneys, eg, segmental atrophy. This negative correlation between hypertension improvement and preoperative status of kidney damage was also apparent in our sample, with a combined hypertension improvement and cure rate at discharge in 86% of our patients with preoperative undamaged kidneys and 38% of patients with damaged kidneys.

Endovascular treatment of RAD seems to be technically possible in only selective cases with limited main stem dissection, and apart from iatrogenic dissection after complicated angioplasty,[27,28] no substantial series are reported in the literature.

CONCLUSIONS

Surgical management of RAD is intended to treat renovascular hypertension and to preserve kidney function. Before planning the operative strategy, careful preoperative examination is necessary to evaluate the exact preoperative status of the diseased kidneys, the extent of RAD, the degree of preoperative kidney damage, and the cause of hypertension. Taking all of our results into account, primary nephrectomy to treat severe renovascular hypertension should be considered only in selective cases, if the dissected kidney is already severely damaged due to infarction, if it has poor function at isotope renography, and if revascularization seems difficult because of renal branch artery involvement. Surgical revascularization of dissected renal arteries is indicated in kidneys with substantial residual renal function and the prognosis is good for permanent hypertension improvement and preservation of kidney function. However, the procedure is technically demanding because of frequent renal artery branch involvement and perivascular scarring.

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